# Physiology of Digestion and Absorption

## Introduction

Digestion begins in the stomach: acid, enzymatic, and mechanical digestion pulverizes the food bolus into chyme. A squirt of chyme gets ejected through the pylorus and into the duodenum. The proximal duodenum senses the chyme's presence and tells the organs of enzymatic digestion—the gallbladder and pancreas—to release their contents—bile and pancreatic enzymes—and flush those contents down their respective ducts with a bicarbonate-rich aqueous fluid. Bile and pancreatic enzymes then chemically digest the chyme's macronutrients down to small polymers. Digestion is completed by the enterocytes, which possess enzymes embedded in the membranes of their microvilli that chemically digest those small polymers down to their monomers. Those monomers have transporters, also located in the microvilli of the enterocytes, that enable the enterocytes to absorb those monomers into the cytoplasm.

You should associate "macronutrients" with the general category of energy source, "smaller polymers" with partially digested macronutrients, and "monomers" with the smallest functional units of that energy source. For example, the protein macronutrient is partially digested into polypeptides (smaller polymers) and fully digested into amino acids (monomers). The fatty acid is the monomer of the triglyceride polymer of the lipid macronutrient. Starch (a carbohydrate) is a long polymer that is partially digested into oligosaccharides (smaller polymers) and, finally, into 6-carbon glucose or fructose monosaccharides (monomers). The meat and potatoes of this lesson are the details of how that meat and potatoes (covered in butter) become amino acids, glucose, and fatty acids in the bloodstream. We then close with mesenteric ischemia, a vascular disorder that commonly affects the segments of absorption.

## The Duodenum Signals for Chemical Digestion

As chyme enters the proximal duodenum, the gallbladder and pancreas need to be told to release their respective juices. The chyme is in the duodenum, so it makes sense for the signal to originate **from the duodenum**. There are multiple sensors and multiple outputs. **Distension** of the duodenum results in a mechanoreceptor-mediated vagovagal response (**neural reflex**). Whereas distension of the stomach stimulates vagus-induced acid secretion (acetylcholine, ACh; gastrin-releasing peptide, GRP), distension of the duodenum does the opposite (inhibits ACh and GRP release in the stomach). More importantly, distension of the duodenum stimulates the vagus nerve to release ACh at the gallbladder (inducing contraction) and pancreatic acinar cells (inducing zymogen release) while also releasing nitric oxide at the hepatopancreatic sphincter (sphincter of Oddi, inducing relaxation). The rest of the duodenal signaling is carried out hormonally by the **enteroendocrine** (entero-, gut; -endocrine, hormones) **cells**, which release **enterogastrones** (hormones). Specifically, we want you to learn **secretin** (stimulate ductal cells to secrete bicarbonate-rich fluid) and **cholecystokinin** (CCK that stimulates zymogen release in the pancreatic acini and smooth muscle contraction in the gallbladder).

| CELL | THEY MAKE | WHAT THEY DO | WHAT STIMULATES THEM |
|---|---|---|---|
| D cells | Somatostatin | "Somatostasis" everywhere, pancreas, GB, stomach<br>↓ Gastric acid | Low pH (like in the antrum) |
| I Cells | Cholecystokinin (CCK) | ↑ Pancreatic acinar secretions<br>↑ GB contraction<br>↓ Gastric emptying | Fatty acids, low pH |
| S Cells | Secretin | ↑ Pancreatic ductal secretion and cholangiocyte secretion of bicarbonate-rich fluid<br>↓ Gastric acid secretion | Fatty acids, low pH |
| K Cells | Gastric inhibitory peptide (GIP) | ↓ Gastric acid secretion | Fatty acids, amino acids |
| Vagus | Vasoactive intestinal peptide (VIP) | Relaxes sphincters | Distension |

**Table 8.1: Duodenal Regulation of Gastric Acid Secretion and the Other Stuff They Do**

This system is immensely complex, and we're going to keep you focused on what you need to know. Vagally mediated acetylcholine makes things "go"—gallbladder contraction and pancreatic secretion. Vagally mediated vasoactive intestinal peptide and nitric oxide relax the hepatopancreatic sphincter. We're going to home in on **I cells** making **cholecystokinin** (CCK) and **S cells** making **secretin**. CCK induces organ action; secretin induces duct action. CCK **induces gallbladder contraction** (releasing bile) and the **pancreatic acinar secretion of zymogens**. Secretin affects the ductal cells, named cholangiocytes in the biliary duct and ductal cells in the pancreas (in this lesson, we simply call them both ductal cells). Secretin induces ductal cells to secrete a **bicarbonate-rich aqueous** fluid.

## Pancreatic Chemical Digestion: Enzyme Physiology

Pancreatic enzymes are necessary to begin chemical digestion, to get those macronutrients down to small polymers. Within pancreatic juice are lipases that digest fats, proteases that digest proteins, and amylases that digest carbohydrates. These enzymes act on all lipids, proteins, and carbohydrates, regardless of where they are and what they are digesting. That means that if these enzymes were active in the acinar cells or pancreatic ducts, they would digest the pancreas. The pancreas must make these enzymes, and get these enzymes into the duodenum, but also ensure that they reach the duodenum before becoming active. So, the question is—how are they kept inactive until they reach the duodenum where they are supposed to act? The answer is complicated but comes down to four mechanisms: zymogens, trypsin inhibitor, ductal secretion, and the location of the activating enteropeptidase.

**Zymogens** (enzyme-ogens) are precursor proteins. They must have inactivating amino acids cleaved off to be activated. All zymogens are enzymes. All enzymes are proteins. Zymogens are named with either a pro- prefix (procarboxypeptidase becomes carboxypeptidase) or an -ogen suffix (trypsinogen becomes trypsin). Pancreatic enzymes can only be activated by **trypsin**. Trypsinogen is a pancreatic zymogen. The thing that activates all the digestive enzymes is packed with the digestive enzymes! Trypsinogen will spontaneously convert to trypsin if not prohibited from doing so. If it does, trypsin activates other trypsins and all the other zymogens prematurely. Thus, trypsinogen must be kept as inactive trypsinogen in the pancreatic ducts (to prevent enzymatic activation too early) but must also be readily activated to trypsin in the duodenum.

Along with the zymogens, acinar cells release **trypsin inhibitor**, which prevents the conversion of trypsinogen to trypsin. Trypsinogen converts to trypsin faster in an acidic environment (such as in the

duodenum, due to the gastric acid and chyme from the pylorus). So, in addition to the trypsin inhibitor, the ductal cells secrete an **alkaline** (bicarbonate-rich) aqueous fluid, which both flushes the zymogens down the ducts and creates an environment that favors trypsinogen remaining trypsinogen. Trypsin will spontaneously convert to trypsin, and the zymogens will be activated. But the alkaline environment, trypsin inhibitor, and rate at which the zymogens are flushed down the ducts mean that the zymogens will get to the duodenum before that happens.

But when the zymogens do get to the acidic duodenum, the activation of trypsin is a good thing. And although those fail-safes protect the pancreas, they hinder the duodenum. So, the duodenum secretes enteropeptidase (formerly enterokinase, but it isn't a kinase, it is a peptidase) into the gut lumen. **Enteropeptidase** cleaves trypsinogen to trypsin, and **trypsin activates everything else**.

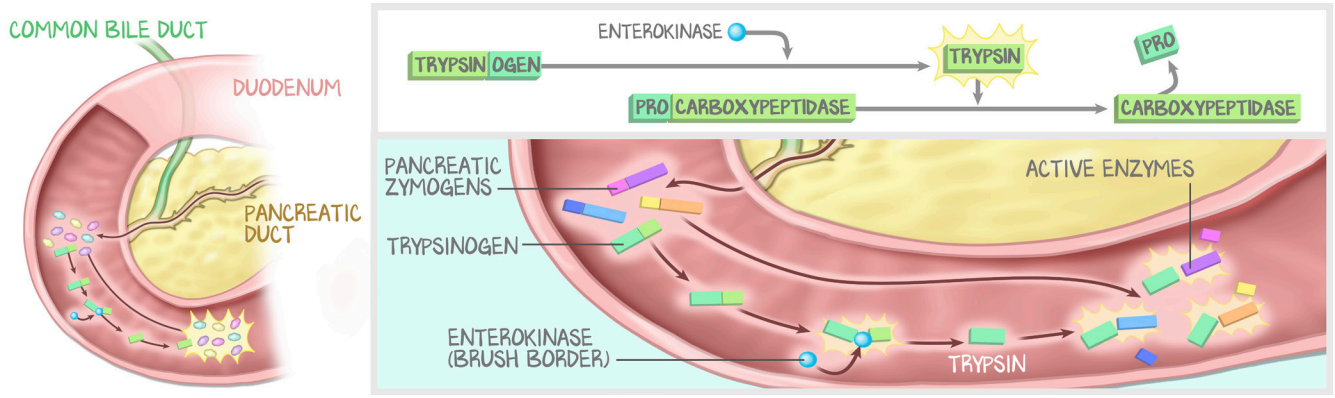


**Figure 8.1: Pancreatic Digestion Physiology—Activation of Zymogens**
Enzymes are released as precursor zymogens. A peptidase activates a zymogen by cleaving off a part of it. Enteropeptidase cleaves trypsinogen to make trypsin. Trypsin is a peptidase that cleaves the other zymogens into their active forms. Active enzymes include proteases, lipases, and amylases.

**Proteases** digest proteins. Chymotrypsin and elastase are **endopeptidases**. They cleave big proteins, somewhere inside the amino acid sequence ("endo-" means cleave "within" the protein), into smaller, more manageable pieces. Carboxypeptidases are **exopeptidases**, chewing off either single amino acids or small blocks of peptide (oligopeptides) and doing so from the carboxyl end of the amino acid chain.

**Pancreatic lipases** digest lipids. A sufficient amount of bile salts (discussed next) must also be present for the digestion of fats. Bile salts emulsify large fat globules into smaller emulsion droplets. Pancreatic lipase acts on these emulsion droplets at the lipid-aqueous barrier, cleaving fatty acids from their glycerol backbone. The lipolytic products—free fatty acids and monoglycerides—are ionized at the duodenal pH of 5.5–6.5 and float away to be absorbed. As they do, new triglycerides emerge from the surface of the emulsion droplet core. As subsequent triglycerides are hydrolyzed, ionized, and float away, the emulsion droplets become progressively smaller, eventually forming micelles. Bile salts then stabilize the fatty acids for absorption by the enterocytes.

**Amylase** digests carbohydrates, specifically large starches. Amylase is released into the acinar ducts in its active form. Amylase hydrolyzes large polysaccharides (macronutrient) to oligosaccharides and large dextrins (small polymers). α-Dextrinase and glucoamylase are luminal enzymes that degrade larger sugars into smaller sugars that the enterocytes can absorb. Associate carbohydrate digestion with amylase and terminal digestion to sugar monomers with "brush border enzymes" (purposefully unnamed).

**CCK** increases the **acinar secretion** of zymogens. **Secretin** increases the **ductal cell secretion** of bicarbonate-rich fluid.

## Gallbladder Chemical Digestion: Bile Salts and Micelles

**CCK** induces **gallbladder contraction** and thus the passage of bile salts into the duodenum. **Secretin** increases **ductal cell secretion** of a bicarbonate-rich fluid. Read the last paragraph of the previous section and this one again. Note their similarities and cross-over.

**Fat globules** are large conglomerations of triglycerides. Bile salts and phospholipids are **amphipathic**, meaning that they have both a hydrophilic end and a lipophilic end. They pick off **emulsion droplets** by tagging the hydrophobic fatty acids with their hydrophobic portions and exposing their hydrophilic portions to the aqueous environment. These are bile-salt-stabilized structures from which individual fatty acids are hydrolyzed away by lipases. The triglycerides don't want to be near water, so they hide in the core of the emulsion droplet. However, on the surface of the emulsion droplet, some triglycerides are exposed to the pancreatic lipases. The free fatty acids that get ionized can be absorbed by the enterocytes. Some of the fatty acids are too big to dissolve in water. As the pancreatic lipases send off the fatty acids and monoglycerides that are small enough to be protonated, the volume of the emulsion droplet decreases.

When all that remain of the original emulsion droplet are cholesterol, fat-soluble vitamins (ADEK), and water-insoluble (i.e., not ionized) free fatty acids and monoglycerides, all stabilized by bile salts, it is called a **mixed micelle**—a mix of bile salts and terminally digested lipids. The mixed micelle approaches the enterocyte microvilli, where **fatty acid transporters** (the details are not important, and their names are confusing) take the cholesterol, vitamins, fatty acids, and monoglycerides from the mixed micelle. Those fatty acids that were ionized and, therefore, not part of the mixed micelle have already been transported into the cytoplasm of enterocytes upstream. This mixed micelle absorption finishes off the absorption of only those lipids that remained stabilized by the mixed micelle. The bile salts of the mixed micelle, now free of their lipid molecules, are dispatched to repeat the process, leaving the enterocyte as a **bile salt micelle.**

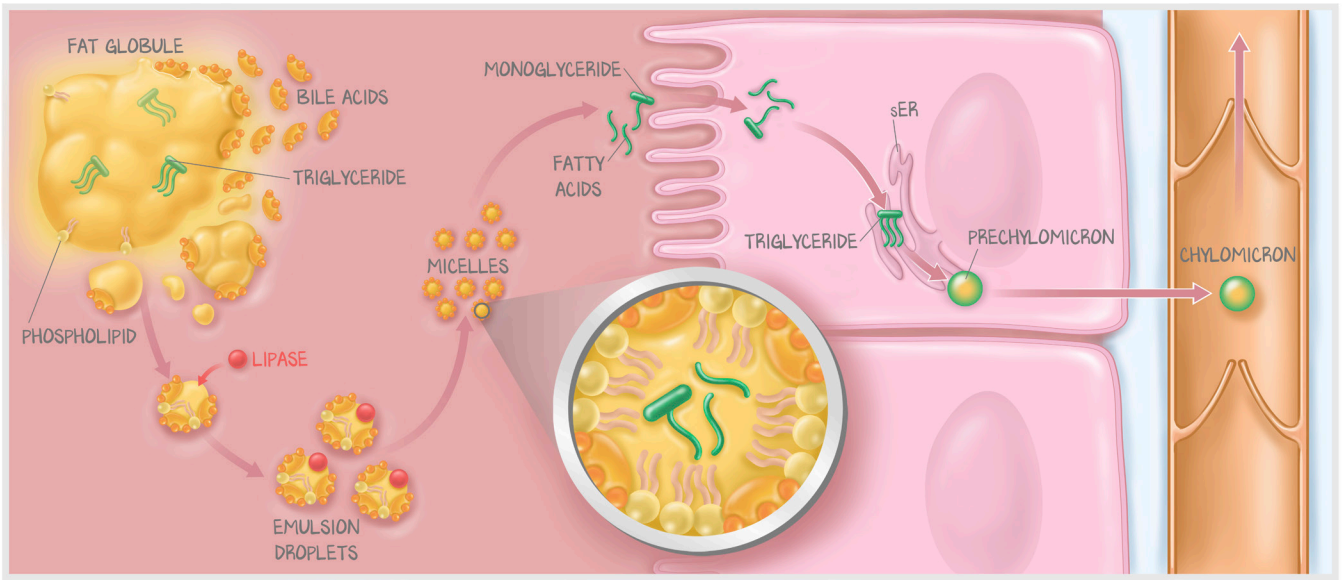


**Figure 8.2: Fat Digestion and Absorption**
Large fat globules are broken down by bile acids to form emulsion droplets. Pancreatic lipase works on the surface of emulsion droplets, cleaving away free fatty acids and monoglycerides. Some of these diffuse to the brush border and are absorbed. Others remain in mixed micelles, the end product of an ever-shrinking emulsion droplet. The mixed micelles also contain fat-soluble vitamins and cholesterol. The mixed micelle enables the absorption of the lipid content. That lipid is packaged into chylomicrons, which are released via exocytosis into the lymphatic vessels of the villi, called lacteals.

Within the cells, the lipid contents are reassembled and stuffed into a lipid-rich vesicle. The triglycerides are assembled from the monoglycerides and free fatty acids in the smooth endoplasmic reticulum. They are passed to the Golgi, from which a vesicle is released. Most of the lipids—now as triglycerides, cholesterol, and fat-soluble vitamins—are stuffed together into a **chylomicron**. This is a lipophilic structure, so it cannot be circulated in the aqueous blood. Chylomicrons are thus released into the **lacteals** of the lamina propria, the **lymphatics**. Glycerol (small) and short-chain fatty acids (also small) pass directly into the portal vein.

## Terminal Digestion and Apical Absorption by the Small Intestine

**Carbohydrate absorption.** Glucose and galactose are absorbed through the **SGLT1** cotransporter. It harnesses the passage of sodium down its concentration gradient to drive glucose or galactose up its concentration gradient into the cell. Fructose is absorbed from the lumen through **GLUT5**. All three monosaccharides are released into the bloodstream by the basolateral **GLUT2** cotransporter. The sodium gradient is established by the $Na^+/K^+$-ATPase on the basal membrane. Only certain **monosaccharides**—glucose, galactose, and fructose—can be absorbed. If any other sugars are present, they will end up in the colon, where bacteria will use them. Brush border intestinal enzymes (not pancreatic enzymes) are responsible for separating disaccharides into their monosaccharide constituents. These enzymes are **sucrase** for sucrose, **maltase** for maltose, and **lactase** for lactose.

**Protein absorption.** Amino acids, dipeptides, and tripeptides are easily absorbed by the brush border. After absorption into the enterocytes' cytoplasm, the dipeptides and tripeptides are further digested into individual amino acids. Only amino acids exit the enterocyte into the portal vasculature. Large peptides are not absorbed. The cell is actually better at absorbing tripeptides and dipeptides than individual amino acids. Not all ingested proteins can be degraded by our intestinal enzymes. Those ingested but not absorbed end up in the colon.

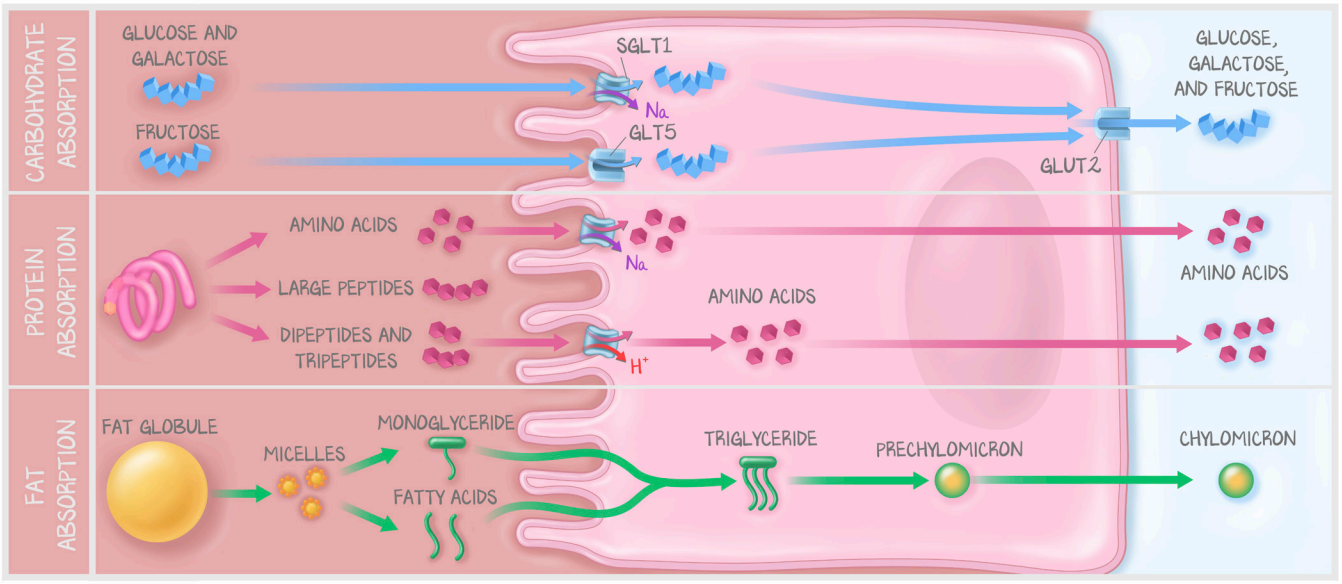


**Figure 8.3: Absorption Summary**
There are three types of energy sources: fat, protein, and sugar. Enterocytes degrade disaccharides into monosaccharides with brush border enzymes and absorb monosaccharides with either SGLT1 or GLUT5. Proteins are degraded into oligopeptides (di- and tripeptides) as well as single amino acids. A sodium/amino acid cotransporter is used to absorb amino acids, and an H+/oligopeptide cotransporter is used to absorb di- and tripeptides, which are degraded into individual amino acids before being released into the bloodstream. Fats are digested from emulsion droplets, their contents absorbed then reassembled before being released into the lymphatics as chylomicrons.

**Fat absorption** was handled in the last section.

## Mechanical Digestion and Peristalsis of the Small Intestine

The intestine does more than just absorb and send signals. Just as the stomach uses churning and peristalsis, so too does the intestine. **Segmentation** (the churning of the small intestine) is the nonpropulsive motion in which segments alternate contraction and dilation. This turns food over, ensuring that the chyme that isn't in contact with the enterocytes (i.e., the chyme in the center of the lumen) is mixed with the chyme that is. Thus chyme is mixed and spun to maximize exposure for absorption. **Peristalsis** is the alternating contraction and relaxation of muscles that propels chyme down the GI tract. Between peristaltic contractions (which will move chyme "one segment down"), intestinal segmentation enables the maximal absorption of chyme wherever its current location may be in the intestine.

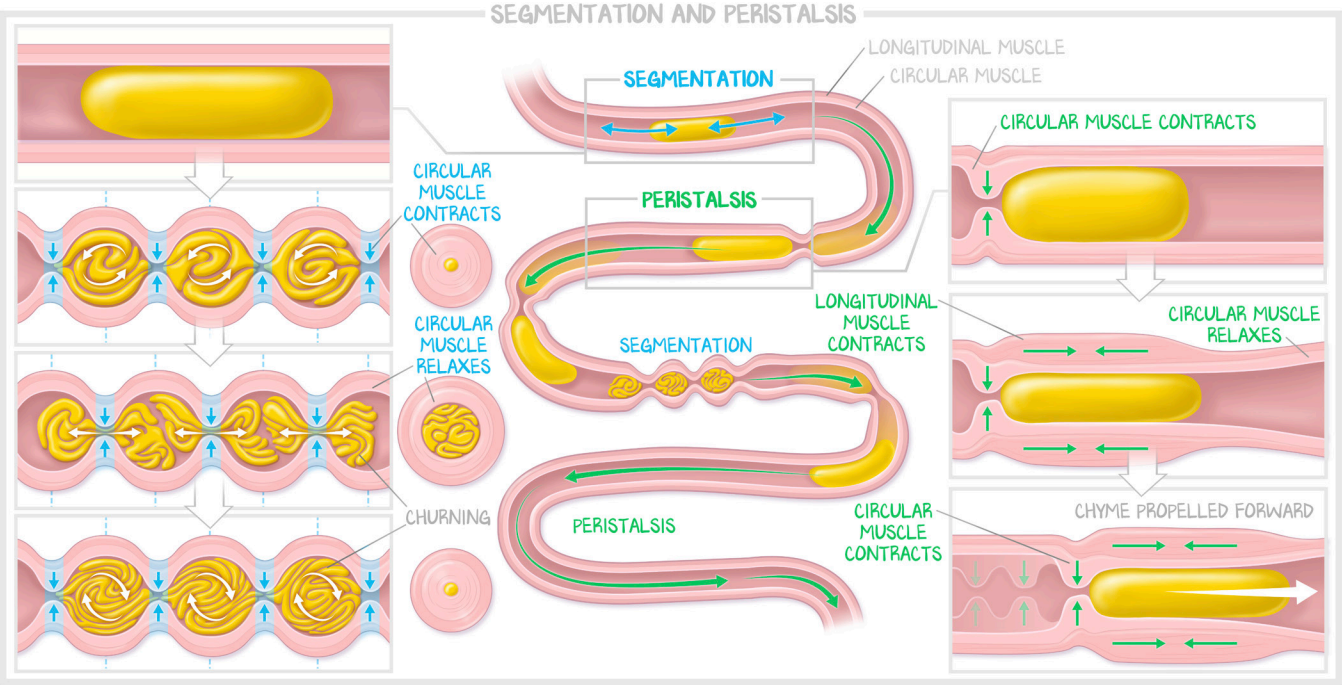


**Figure 8.4: Segmentation and Peristalsis**
Segmentation is the contraction and relaxation of circular muscles between peristaltic contractions. This mixing enables the chyme to be turned, mixed, and churned to ensure that the contents at the center of the lumen come into contact with the brush border. The goal is to ensure that the epithelium has as much contact with the chyme as possible. Then peristalsis enables propulsive movement forward. Behind the bolus, the circular muscles are the most contracted, and the longitudinal muscles are relaxed. In front of the bolus, the circular muscles are the most dilated, and the longitudinal muscles are the shortest they can be, the most contracted they can be. Farther down, the circular muscles dilate, and with that dilation, the longitudinal muscles contract.

## Where Absorption Occurs

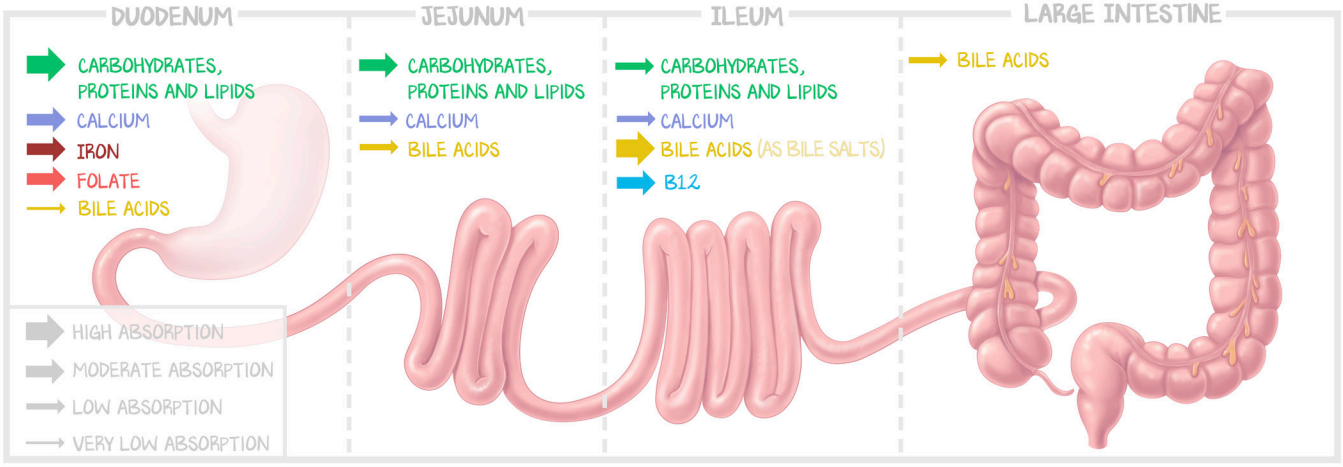


**Figure 8.5: Site of Absorption**
The number of loops of bowel is used to represent how far along the GI tract the structure is. The magnitude of the arrow represents how much is absorbed at that site as compared to other sites, not to the other nutrients. Most important are FIC (duodenum only) and the terminal ileum (bile salt reabsorption).

Because the concentration of stomach contents is greatest as soon as they leave the pylorus, the most absorption occurs in the duodenum, even though the jejunum is ideally suited for absorption histologically. The proximal duodenum digests, the distal duodenum absorbs. The rest of the small intestine absorbs. **Carbohydrates, proteins,** and **lipids** are absorbed throughout the GI tract. **Folate and iron** are absorbed **only** in the duodenum. Although calcium is absorbed throughout the entire small intestine, the primary location of absorption is in the duodenum, so we teach that the FIC minerals (folate, iron, calcium) are absorbed by the duodenum. **Bile salts** are actively absorbed in the ileum, and some bile salts become bile acids and are absorbed passively (see GI: Hepatobiliary #2: *Physiology of Bile and Bilirubin*). **$B_{12}$** is also absorbed in the terminal ileum, and only in the presence of intrinsic factor (see Heme/Onc: Anemia #6: *Macrocytic Anemia*).

## The Different Segments of the Small Intestine

The small bowel is the entire length of the intestines between the stomach and the colon. It is long, twisting and turning beneath the stomach and the greater omentum. It is broken up into three functional segments: the **duodenum** or proximal segment, the **jejunum** or middle segment, and the **ileum**. The sections cannot be differentiated simply by looking at them with the naked eye, except in the abdomen, where their location relative to the stomach (duodenum) or colon (ileum) implies which segment they most likely are. Instead, the segments are distinguished by their histology. The transition is gradual and unlike the GE junction and pectinate line, which have abrupt changes in the epithelium that can be demonstrated on the same histology slide. The function, and therefore histology, of the

terminal duodenum resembles that of the early jejunum, and the early ileum resembles the terminal jejunum. Even though it is more continuous than segmented, we teach it in segments for clarity.

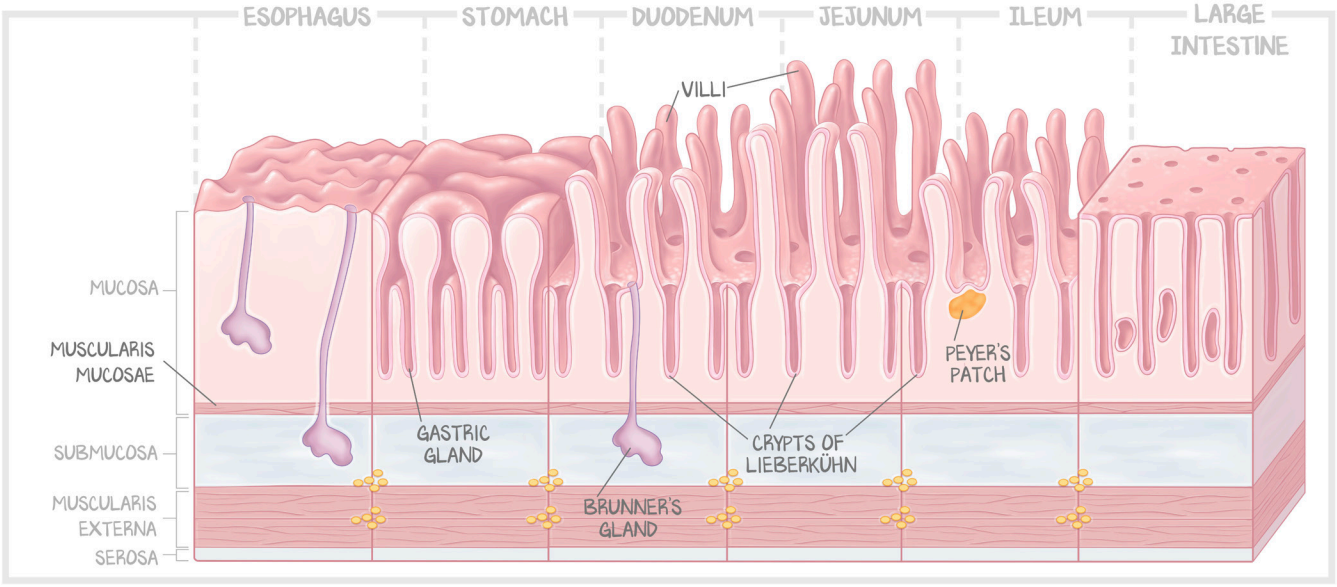


**Figure 8.6: Regional Differences of the Mucosa**
From stomach to colon, the epithelium is simple columnar. The mucosa is made complex by invaginations of that simple columnar epithelium into the lamina propria. These invaginations are called glands in the stomach and crypts everywhere else. Deep in the crypts (in the intestines, called crypts of Lieberkühn) are epithelial stem cells. In addition, the small intestine mucosa has evaginations into the lumen called villi. On the surface of those villi are enterocytes. At the tips of the enterocytes are microvilli. What separates the segments of the small intestine are the appendages.

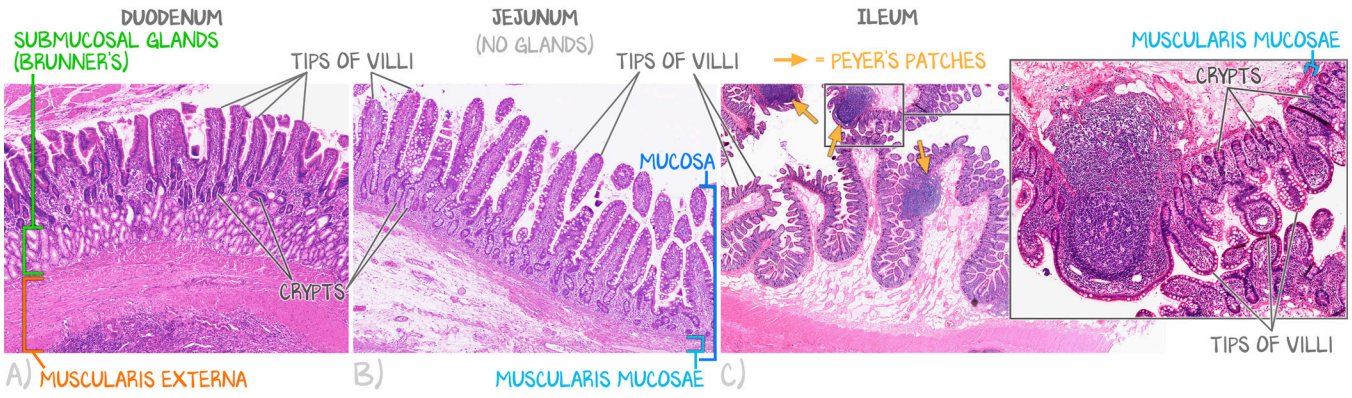


**Figure 8.7: Specific Histology of Each Segment of Small Bowel**
(a) Intermediate-magnification view of the duodenum showing villi, Brunner's glands, and muscularis propria. Brunner's glands appear as a band of round, oval, or tubular glands with an epithelium with light pink cytoplasm sandwiched between the villi above and muscularis externa below. (b) Intermediate-magnification view of the jejunum. The mucosa is folded into long, delicate, finger-like villi that have a lamina propria core. The villi are covered with columnar epithelium and numerous goblet cells. A row of intestinal glands (crypts) can be seen at the base of the villi. There are no Brunner's glands (seen in the duodenum) or lymphoid aggregates (Peyer's patches, seen in the ileum) in the jejunum. (c) High-magnification view of the ileum. As compared to those in the duodenum and jejunum, the villi in the ileum are shorter and more finger-like in shape and have a greater proportion of goblet cells within the epithelium. Another characteristic histologic feature of ileum is the presence of specialized lymphoid aggregates called Peyer's patches. They are in the mucosa, though they appear to spill into the submucosa as the muscularis mucosae is harder to see when the lymphocytes proliferate so much and so quickly.

**Appendages** refers to the unique characteristics of a given segment's histology. The duodenum has **Brunner's glands**, bicarbonate-secreting glands in the **submucosa**. The esophagus and duodenum are

the only locations with submucosal glands and the only locations outside the stomach that experience gastric pH. If there are villi, it is intestine. If there are villi and submucosa filled with circular-shaped structures lined with cells, it is the duodenum. In the ileum, **Peyer's patches**, lymphoid aggregates that resemble germinal centers, are found in the lamina propria. The jejunum has no special structures to define it—it lacks Brunner's glands and Peyer's patches. But what most distinguishes it is that it lies between digestion in the duodenum and infection in the colon. It has a perfect environment to do one thing: absorb. And so, the villi are the tallest, the microvilli the most abundant, and because it is dedicated to nothing but absorption, it has the fewest extraneous types of cells.

## Intestinal Vascular Pathologies

There is a progression in the anastomoses (arterial arcades) proximal to the terminal branches (vasa recta) that feed a given segment of bowel from foregut to hindgut. The **arterial arcades** are conglomerations of redundant vasculature. The terminal branches of these arcades each perfuse a specific region of the intestine. These terminal branches are called **vasa recta**. The duodenum and jejunum have long vasa recta and short arterial arcades. The ileum and colon have shorter vasa recta because there are longer arterial arcades. Both arrangements ensure that a blockage in the medium and small arteries will not cause ischemic injury as there is plenty of collateral circulation around the arcade. The implication of this is that **embolization can block vasa recta**, but **atherosclerosis must occur at the aortic branch** to provoke symptoms. That gives us chronic mesenteric ischemia and acute mesenteric ischemia.

**Chronic mesenteric ischemia.** Atherosclerosis can occur in any vessel. When atherosclerosis (Cardiology: CAD #1: *Pathophysiology of Atherosclerosis*) gets bad enough, usually around 70% stenosis, symptoms are provoked. Those symptoms are determined by the organ distal to the atherosclerotic artery. When we discussed ischemic insult to the heart, the chest pain it caused was called angina. If demand increased (exercise), angina struck. In parallel, if the superior mesenteric artery has reached or exceeded critical stenosis (70% occluded), then when the demand of the intestines increases, it can provoke "intestinal angina." The occlusion prevents the delivery of enough blood to meet the demands of the intestine. If the work is decreased, symptoms abate. Intestine works harder when it has to absorb food. So, chronic mesenteric ischemia presents with **pain after eating**, and subsequent **avoidance of eating** leads to **weight loss**. The patient learns that eating provokes pain, so does not eat. Chronic mesenteric ischemia is "stable angina" of the intestine. Atherosclerosis is most likely found at the superior mesenteric artery. The celiac trunk is rarely affected.

**Acute mesenteric ischemia.** If chronic mesenteric ischemia is the "stable angina" of the intestine, then acute mesenteric ischemia is the "acute coronary syndrome" of the intestine. Caused by either **plaque rupture with thrombus formation** (atherosclerosis) or **embolization of a thrombus** (such as in AFib or atherosclerosis), the result is total occlusion of a mesenteric artery. The arcades ensure that there is redundant collateral circulation within the mesentery. But if there is a thrombus of the common artery that feeds the entire arcade, that redundancy doesn't matter. In mesenteric ischemia, bowel dies. Dying bowel hurts. The tissue farthest from the arterial occlusion dies first. The orientation of the vasa recta is to penetrate the gut tube from the serosa (the outside) towards the mucosa. The mucosa is the farthest thing from the arterial supply, so it dies first. Because it dies first, there may not be inflammation of the exterior of the bowel. That means there is no inflammation of the peritoneum of the abdominal wall, so there will be no peritoneal signs. The sign of vascular abdominal pain is **pain out of proportion** to the physical exam. The patient is in pain. The lactic acid is through the roof (a sign of anaerobic metabolism and poor tissue perfusion). The patient is floridly septic—fever, tachycardia, leukocytosis. But the **abdomen is soft**. At least until the bowel perforates and the patient becomes peritoneal. After the bowel dies, two things happen. The first is that the dead bowel stops moving, which presents with **decreased**

**bowel sounds**. The second is that the dead bowel sloughs off, presenting with **bloody diarrhea**. These are both symptoms that the bowel is long dead. Better to catch it first with an angiogram and either resect the dying bowel or alleviate the obstruction.

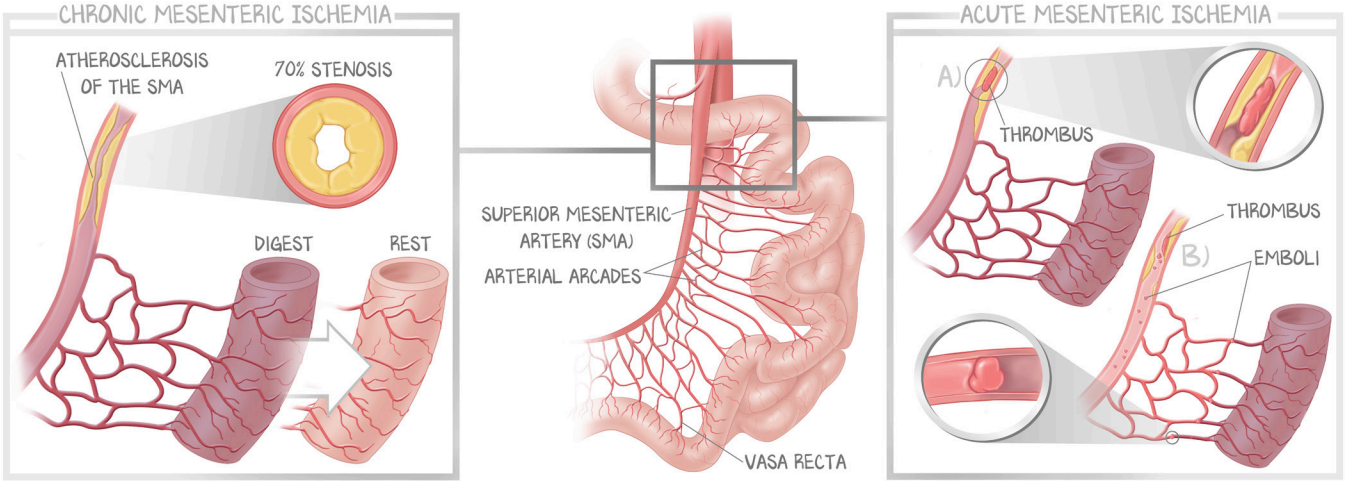


**Figure 8.8: Mesenteric Ischemia**
Because the arterial arcades allow for so many anastomoses, the only way to cause ischemia of the bowel is for a blockage to be proximal to the arcades or distal to the vasa recta. As it turns out, those are essentially the same disease process. When the superior mesenteric artery develops a critical stenosis (70% occlusion), symptoms may arise. When demand is increased—when food is digested and absorbed—there will be ischemic symptoms (pain). But that critical stenosis could also undergo rupture and thrombosis, leading to ischemia of that artery's territory, acute mesenteric ischemia. Like with a myocardial infarction and heart muscle, the thrombosis must be opened if the bowel is to survive. Another consequence of that critical stenosis could be thrombosis and embolism, the individual emboli are too small to cause a significant loss of vasa recta, but together, the showering of emboli compromise enough vasa recta to cause serious damage.

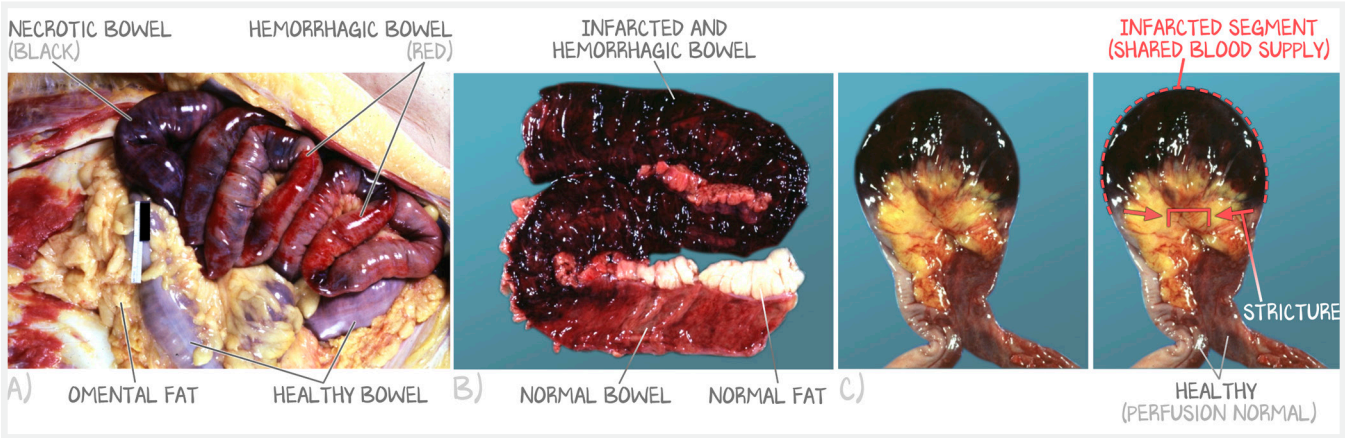


**Figure 8.9: Acute Mesenteric Ischemia**
(a) Autopsy photograph of dead (necrotic), dying with perfusion restored (hemorrhagic), and normal bowel, still within the patient and covered in omental adipose. (b) Removed and dissected sample before formalin fixation (so you see the natural color) showing the necrotic and hemorrhagic infarcted bowel. There is a clear demarcation between the dead bowel and normal bowel, indicating the arrangement of the arterial arcades. (c) Before dissection, the necrotic segment can be seen with normal bowel on either side. The necrotic area had its blood supply compromised, whereas the other segments were unaffected.

## Citations

Figure 8.7: Courtesy of WebPathology.com.

Figure 8.9: Originating from the University of Alabama at Birmingham, Department of Pathology PEIR Digital Library at http://peir.net pursuant to a license grant by the UAB Research Foundation.